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Attenuation of Pressor Response to Laryngoscopy and Endotracheal Tube Intubation with Dexmedetomidine

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Abstract **Original Research Article**

To study the effectiveness of Dexmedetomidine in the dose of 1 µg/kg to attenuate the pressor response to laryngoscopy and endotracheal intubation, 100 patients posted for elective surgery under general anesthesia were selected for this double blinded prospective study with inclusion and exclusion criteria and after institutional ethical committee approval and informed consent from the patients, the eligible patients were divided into two groups 50 in each. Group(S)-Received normal saline and 10 minutes before the induction, the patients were given 100ml of NS over 15 minutes. Group (D)-Received dexmedetomidine and 10 minutes before the induction, the patients were given Inj.Dexmedetomidine 1 µg/kg in 100 ml of normal saline over 15 minutes. After receiving the drug, patients were monitored. All the patients were premedicated and same anaesthetic agents were used for conduction of general anaesthesia in all patient. Laryngoscopy was performed gently by an experienced anaesthesiologist in less than 15 seconds and intubated while monitoring the blood pressure, pulse rate, and E.C.G. This study was concluded that patients in Group-D (Dexmedetomidine) had better control of hemodynamic changes during laryngoscopy and endotracheal intubation than the Group-S(Saline) without any side effects.

Key words: General anaesthesia, Dexmedetomidine, Saline, laryngoscopy, endotracheal intubation, pressor response.

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INTRODUCTION

The sympathetic reaction is activated by a vast range of stressful stimuli including both psychological and physical stresses. Afferent neurons of the sympathetic system are multiple. The major source of excitatory drive to sympathetic preganglionic neurons comes from the rostral ventrolateral medulla. This region of brain stem contains the cardiorespiratory and vasomotor autonomic control centre[1]. It is undisputable that the adrenergic reaction is crucial to survive the insult of major trauma. However in critical illness, an overshooting influence of the sympathetic system can become hazardous and several organ systems may be affected. The effects include increased blood pressure, tachyarrythmias and ischaemic changes in cardiovascular system, increased intracranial tension, increased cerebral blood flow and cerebral haemorrhage in central nervous system, increased oxygen consumption, hyperlactatemia and hyperglycaemia in metabolism, pulmonary oedema and increased pulmonary artery pressure in respiratory system. Among these, the cardiovascular system seems to be most susceptible to sympathetic over stimulation.

Laryngoscopy, endotracheal tube intubation and other airway manipulations are noxious that stimulate sympathetic activity. The cardiovascular response to this noxious stimulus is initiated by proprioceptors responding to tissue irritation in the supraglottic region and in the trachea, located in close proximity to the airway mucosa. This proprioceptor consists of mechanoreceptors with small diameter myelinated fibers, slowly adapting fibers and polynodal endings of nonmyelinated nerve fibres. The superficial location of these proprioceptors and the nerve fibres explains why the topical local anaesthetic applied in the airway is such an effective means of blunting the cardiovascular responses to the airway irritation. The Vagus and the glossopharyngeal nerves carry the afferent stimuli from epiglottic region and activate the vasomotor centre to cause a peripheral sympathetic adrenal response to release adrenaline and nor-adrenaline. In infants and small children, it manifests as bradycardia but in adults, the most common response to

airway manipulation is hypertension and tachycardia. In normal patients, the transient increase in sympathetic activity is tolerated but in susceptible individual like patients with cardiac disease, hypertension, cerebrovascular disease, and hyperthyroidism, it evokes life threatening conditions as mentioned above. Myocardial ischemia might occur during the laryngoscopy and intubation in patients with coronary artery disease. Intraoperative ischemia has been associated with a high rate of perioperative myocardial infarction.

Various methods have been used to attenuate the hemodynamic response to laryngoscopy and ET tube intubation. Examples are shortening the duration of laryngoscopy, narcotics, beta blockers, vasodilators, calcium channel blockers, nerve blocks etc. Studies have already been available with Dexmeditomidine with different doses. Our study was done to evaluate the effectiveness of Dexmedetomidine in the doses of 1 µg/kg to attenuate the pressor response to laryngoscopy and endotracheal intubation

Aim and objectives

To evaluate the effectiveness of Dexmedetomidine as single premedicant dose to attenuate the pressor response to laryngoscopy and endotracheal intubation

The following parameters were studied

- Heart rate
- Systolic Blood pressure
- Diastolic Blood pressue
- Mean arterial Blood pressure
- Any complications during the study

MATERIAL AND METHODS

This was a double blinded prospective study conducted in Dhanalaksmi Srinivasan Medical College, Perambalur, and Tamil nadu in the year of 2017. After institutional ethical committee approval, a total of 100 patients belonging to ASA-I category posted for elective surgery under general anesthesia were enrolled. The inclusion criteria were a) 20-50 years of either sex b) ASA-I category patients and the exclusion criteria were a) patient with refusual of consent b) Cardiovascular disease c) Cerebrovascular disease d) Hepatic dysfunction e) Pregnancy f) Patients with psychiatric disease g) Hyperthyroidism h) patients with beta blocker therapy i) patients with alcoholic dependence. Pre-anesthetic work up was carried out in anaesthesia assessement clinic for all the patients and Informed consent obtained. All patients were given Tab. Alprazolam 0.5mg orally on the night before surgery and they were fasted for 8 hours. The basal vital parameters were recorded on the morning day of the surgery. Eligible patients were divided into two groups 50 in each. Group(S) was considered as control group and 10 minutes before the induction, the patients were given 100ml of normal saline infusion over 15 minutes. Group (D) was considered as study group and 10 minutes before the induction, the patients were given Inj. Dexmedetomidine 1 µg/kg (in 100ml of normal saline) over 15 minutes. After receiving the drug, patients were monitored.

All the patients were premedicated with inj. Glycopyrrolate 0.2 mg intravenously. After the preoxygenation, patients were induced with Profopol 2mg/kg and Laryngoscopy was performed gently by an experienced anaesthesiologist in less than 15seconds after succinyl choline 2mg/kg intravenouly and intubated while monitoring blood pressure, pulse rate, and E.C.G. The patients requiring laryngoscopy >15 seconds, bronchospasm, laryngospasm were eliminated from the study. Vitals were monitored over the period of 10 minutes as following

T_1-Basal reading,
T_2-On induction
T_3-On laryngoscopy and intubation
T_4-After 1minute of intubation
T_5-After 3 minutes of intubation
T_6- After 5 minutes of intubation
T_7- After 10 minutes of intubation

The maintenance of anesthesia was done with 2:1 (N_2O/O_2), fentanyl, vecuronium and sevoflurane. Residual neuromuscular blockade was reversed with neostigmine and glycopyrrolate. After complete recovery, all patients were extubated and observed in post anaesthesia care unit. Vitals were monitored. Results were presented as mean ± Standard Deviation and categorical data were presented in number and percentage. Statistical analysis was done using Medcalc software and P value <0.05 was taken as statistically significant.

RESULTS

Demographic Data (Table-1&2)

Table-1: (Gender)

Gender	Control group	Study group
Male	27(54%)	25(50%)
female	23(46%)	25(50%)
Total	50(100%	50(100%)

Table-2: (Age group)

Age group	Control group (N=50)	Study group (N=50)
20-30years	12(24%)	11(22%)
31-40years	20(40%)	19(38%)
41-50years	18(36%)	20(40%)

OBSERVATION

Dexmedetomidine group had better control of heart rate 62.21± 7.27 compared to saline group 78.54±12.61 with statistically significant p value of less than 0.0001.

Table-3: (Mean Pulse rate with standard deviation)

TIME	Control group	Study group	P-VALUE
T_1	80.32±7.72	81.12±8.47	0.6227
T_2	83.02±11.35	76.30±13.61	0.0004
T_3	78.54±12.61	62.21±7.27	<0.0001
T_4	93.32±10.01	68.50±13.90	<0.0001
T_5	105.76±9.58	70.22±14.19	<0.0001
T_6	87.50±11.20	70.52±13.81	<0.0001
T_7	86.50±11.15	71.44±13.91	<0.0001

Mean systolic pressure was also significantly attenuated during the critical procedure of GA in Dexmedetomidine group 109.46±10.56 compared with saline group 133.98±10.57 with statistically significant p value of less than 0.0001

Table-4: Mean systolic blood pressure with standard deviation

TIME	Control group	Study group	P-VALUE
T_1	118.82±8.59	116.82±8.69	0.2500
T_2	117.80±9.38	108.80±10.24	0.0001
T_3	133.98±10.57	109.46±10.56	<0.0001
T_4	131.40±11.74	108.609±10.24	<0.0001
T_5	119.91±8.01	106.75±6.21	<0.0001
T_6	117.96±7.77	108±10.64	<0.0001
T_7	116.60±7.79	110±10.21	<0.0001

Mean diastolic blood pressure was well in control in dexmedetomidine group 62.58±12.89 compared to saline group 73.28±9.54 with statistically significant p value of less than 0.0001.

Table-5: Mean diastolic blood pressure with standard deviation

TIME	Control group	Study group	P-VALUE
T_1	69.6±8.401	72.80±9.17	0.01719
T_2	75.24±9.40	66.06±10.61	0.0005
T_3	73.28±9.54	62.58±12.89	<0.0001
T_4	74.68±13.96	61.59±12.28	<0.0001
T_5	79.16±12.99	61.27±12.18	<0.0001
T_6	81.16±12.35	59.12±11.59	<0.0001
T_7	82.82±12.01	59.35±11.35	<0.0001

Mean arterial pressure was also in control in dexmedetomidine group 77.98±7.86 compared to saline group 88.12±7.75 with statistically significant p value of less than 0.0001. In the entire group no complications were noted

Table-6: Mean arterial pressure with standard deviation

TIME	Control group	Study group	P-VALUE
T_1	86.00±8.50	87.20±9.02	0.5027
T_2	86.6±9.62	83.64±8.	0.1048
T_3	88.12±7.75	77.98±7.86	<0.0001
T_4	94.45±6.20	77.54±8.29	<0.0001
T_5	96.57±8.46	77.07±7.950	<0.0001
T_6	94.08±9.42	75.39±8.46	<0.0001
T_7	94.53±11.07	75.57±10.27	<0.0001

DISCUSSION

Most of the general anesthesia requires endotracheal intubation especially surgical procedures involving the head and neck, thorax, and abdomen. Laryngoscopy and endotracheal intubation are considered to be the most critical procedure of General anaesthesia in which it provokes transient but marked sympathetic/sympathoadrenergic response. Studies had already been done using various agents but they have their own limitations like use of halothane associated with dysrhythmias, reflex tachycardia in calcium channel blockers and vasodilators, respiratory depression, chest wall rigidity causing difficulty in ventilation and post operative nausea and vomiting in opioids. Dexmedetomidine is an alpha$_2$ agonist like clonidine with high selectivity for alpha$_2$ receptors with lack of respiratory depression. Alpha$_2$ agonists clonidine and dexmedetomidine have already been used successfully in previous studies in attenuating the pressor response induced by laryngoscopy and endotracheal intubation. The effects of dexmedetomidine on blood pressure is biphasic with an initial transient rise of blood pressure and heart rate brought about by stimulation of alpha$_2$ B subtype receptors found in vascular smooth muscle. This is followed by fall in blood pressure and heart rate due to the feedback inhibition [6] of sympathetic outflow with stimulation of pre synaptic alpha$_{2\ A}$ receptors leading to inhibition of release of noradrenaline. The baroreceptor reflex is well preserved with the use of dexmedetomidine and the bradycardia is also easily treatable, confirming the hemodynamic stability. Direct laryngoscopy and endotracheal intubation following induction of anesthesia is associated with hemodynamic changes due to reflex sympathetic discharge caused by epipharyngeal and laryngopharyngeal stimulation. This increased sympatho-adrenal activity may result in hypertension, tachycardia and arrhythmias [3, 4]. This increase in blood pressure and heart rate are usually transient, variable and unpredictable. Transient hypertension and tachycardia are probably of no consequence in healthy individuals, but either or both may be hazardous to those with hypertension, myocardial insufficiency and cerebrovascular diseases [5-7]. At least in such individuals there is a necessity to blunt this response.

Dexmedetomidine produces hemodynamic stability by altering the stress-induced sympathoadrenal responses to intubation during surgery and during emergence from anesthesia [7]. Scheinin *et al*. [8] studied the effect of dexmedetomidine on tracheal intubation, required dose of induction agent and preoperative analgesic requirements and they concluded that the required dose of thiopentone was significantly lower in the dexmedetomidine group and the drug attenuated the hemodynamic responses to intubation. Jaakola *et al*. [9] in their study concluded that dexmedetomidine attenuated the increase in heart rate and blood pressure during intubation. Raj Bahadur Singh [11] conducted comparative study of dexmedetomidine and diltiazem for attenuating pressor responses to laryngoscopy and endotracheal intubation and concluded that both the drugs were safe and effective in attenuating the hemodynamic response following laryngoscopy and endotracheal intubation; however, between two trial drugs, dexmedetomidine had a better response. Vinit Kumar Srivastava [12] *et al*. did a Comparative evaluation of esmolol and dexmedetomidine for attenuation of sympathomimetic response to laryngoscopy and intubation in neurosurgical patients and concluded Dexmedetomidine 1 µg/kg was more effective than esmolol for attenuating the hemodynamic response to laryngoscopy and intubation in elective neurosurgical patients. Bon Sebastian *et al*. [13] compared between two doses of dexmeditomidine and they were effective for Attenuation of haemodynamic responses to laryngoscopy and endotracheal intubation**.** Sulaiman S *et al*. [14] concluded that Dexmedetomidine can be considered before induction of general anesthesia in patients undergoing myocardial revascularization, even if the patients are receiving beta blockers.

In our study hemodynamic parameters like heart rate, systolic blood pressure, diastolic blood pressure and mean arterial pressure were well in control in dexmedetomidine group during laryngoscopy and endotracheal intubation in comparison with saline group. Our results were in accordance with the previous study [8-15]. Usage of dexmedetomidine also produced arousable sedation without airway compromise and no side effects.

CONCLUSION

Study was concluded that dexmedetomidine with the dose of 1µg/kg of body weight attenuated the pressor response induced by laryngoscopy and endotracheal intubation without any side effects. Hence it can be used safely.

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